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Do Red Herrings Swim in Circles?

Controlling for the Endogeneity of Time to Death

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Do Red Herrings Swim in Circles? – Controlling for the Endogeneity of Time to Death

Abstract
Studies on the effect of ageing on health care expenditures (HCE) have revealed the importance of controlling for time-to-death (TTD). These studies, however, are subject to possible endogeneity if HCE influences remaining life expectancy. This paper introduces a ten year observational period on monthly HCE, socioeconomic characteristics, and survivor status to first predict TTD and then uses predicted values of TTD as an instrument in the regression for HCE. While exogeneity of TTD has to be rejected, core results concerning the role of TTD rather than age as a determinant of HCE (the "red herring" hypothesis) are confirmed.

JEL Classification: I10, D12

Keywords: Health care expenditure, proximity to death, ageing, "red herring" hypothesis

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1. Introduction

This paper seeks to add to the debate on the 'red herring' hypothesis, viz. the claim that population ageing will not have a significant impact on health care expenditure (HCE) (*Zweifel* et al., 1999). Several authors (*Salas* and *Raftery*, 2001, *Seshamani* and *Gray*, 2004) disputed the robustness of these findings, pointing to potential weaknesses in the econometric methodology. Their main arguments referred to multicollinearity between and endogeneity of the explanatory variables. Use of the Heckman model to deal with the fact that HCE are censored, zero-inflated, and roughly log-normally distributed may run into multicollinearity problems because the inverse Mill's ratio λ is often highly correlated with the other explanatory variables. Regarding endogeneity, *Salas* and *Raftery* (2001) argued that time-to-death (TTD) is influenced by current and previous HCE, causing OLS estimates to be biased and inconsistent.

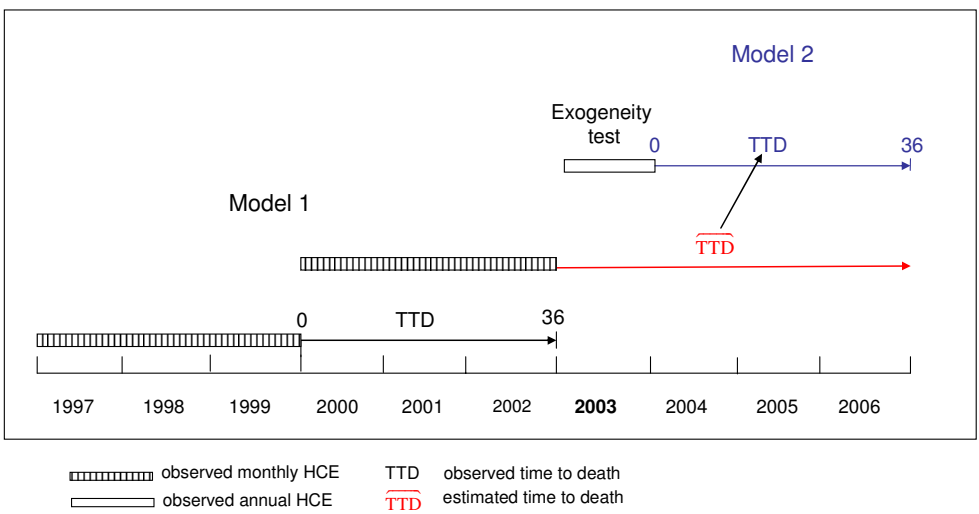
These methodological concerns were addressed in *Zweifel* et al., 2004) and *Werblow* et al., 2007). Multicollinearity was at least mitigated by employing a two-part model in addition to the Heckman model. The two-part model separates the selection part (probability of positive HCE) from the equation for the level of HCE, serving to eliminate the correlation between the selection term λ and the other regressors as a source of multicollinearity. As to endogeneity, the solution adopted was to reduce the number of TTD indicators that may be influenced by HCE. Rather than using a panel data set with quarterly HCE up to the time of death for each deceased [as in *Zweifel* et al., 1999), where a set of potentially endogenous quarter dummies may cause problems], HCE of one year only was related to TTD.

In this paper, the panel data set covers the years 1997 to 2006, permitting to use the first three years of monthly HCE and observed TTD values up to the end of 2002 to derive estimated TTD values for the 2003 to 2006 period (see *Figure 1*). With data of about 60,000 individuals of whom 11 percent died between 2000 and 2006, endogeneity of TTD can directly be tackled in this way.

Accordingly, this paper is structured as follows. In section 2, monthly HCE covering 1997 to 1999 are related to TTD of individuals who died between 2000 and 2002 or survived December 31, 2002 (Model 1, with maximum TTD set at 36 months; see *Figure 1* again). Estimated coefficients then serve to calculate estimated TTD ($\widehat{TTD}$) from individuals' monthly HCE between 2000 and 2002. This variable serves as the main instrument in the instrumental variable (IV) estimation of model 2. In Section 3, annual HCE in 2003 is analyzed as a function of, age, TTD and other variables. In this model 2, maximum TTD is 36

months, attributed to a person who survived December 31, 2006. To avoid endogeneity, TTD values derived from model 1 enter the regression as instrument. Since the pertinent tests point to endogeneity of TTD, the question arises of whether the 'red herring' hypothesis is robust with regard to the measurement of time-to-death. This issue is taken up in Section 4, which presents a regression for HCE in 2003 that contains expected TTD estimated from past HCE and demographic factors but no survival information. According to our findings, time-to-death continues to be a highly significant determinant of HCE, causing its age gradient to even become negative past the age of 60 at the latest. Section 5 summarizes and concludes.

Figure 1: Setup of the study



2. Estimating TTD using monthly HCE covering three years

Model 1 considers 8,650 individuals with residence in the Swiss cantons of Zurich and Geneva, of which 3,124 died between 2000 and 2002 and 5,526 survived to the end of 2002 (see *Table 1*). Survivors represent a 10 percent sample randomly drawn from the population. Average TTD of the deceased is about 18 months; for the survivors, TTD is set to 36 months. The average age in 1999 of the deceased is 76 years, that of the survivors, 55 years. There is a 3 percentage point larger share of men among the deceased than among the survivors,

reflecting their lower life expectancy. Zurich's share is higher among the survivors than among the deceased, indicating a younger population in Zurich than in Geneva.

Swiss social health insurance law allows individuals to choose higher deductibles. Among the deceased, one-fifth opted for high-deductible contracts, compared to one third among the survivors. Moral hazard effects not only emanate from medical insurance but also from other insurance coverage linked to health status (*Zweifel* and *Manning*, 2000). Notably, generous income replacement in the event of sickness or accident serves to further mitigate the financial consequences of an illness episode. For this reason, accident insurance is taken into account as well (to the extent that it is bought from the same health insurer). This option is often chosen by the elderly, while individuals in the labor force usually obtain accident insurance through their employer. This explains why the share of individuals combining health and accident insurance is much higher among the deceased. Roughly one-third of the insured opted for hospital supplementary insurance providing for amenities. Between 84 and 94 percent chose at least one additional supplement to their health insurance policy (not detailed in *Table 1*). These supplements are also expected to give rise to (more limited) moral hazard effects.

Table 1: Descriptive statistics I, 2000

	Deceased (n = 3,124)				Survivors (n = 5,526)			
Variable	**Mean**	**SE**	**Min**	**Max**	**Mean**	**SE**	**Min**	**Max**
Age	76.38	13.37	30	95	55.35	15.1	30	95
Time to death in month	18.07	10.35	1	36	36.00	0	36	36
Share of men	0.43	0.50	0	1	0.40	0.49	0	1
Share of individuals from Zurich	0.67	0.46	0	1	0.78	0.41	0	1
Share of individuals								
with higher deductibles	0.21	0.41	0	1	0.41	0.49	0	1
with accident insurance	0.93	0.25	0	1	0.68	0.47	0	1
with suppl. hospital insurance	0.32	0.47	0	1	0.44	0.50	0	1
with other supplements	0.84	0.37	0	1	0.94	0.24	0	1

Table 2 compares monthly HCE between deceased and surviving persons in the years 1997 to 1999.[1] The average HCE over the 36 months was 950 Swiss Francs (CHF, some US$ 630 at 2006 exchange rates) for the deceased and 247 CHF for the survivors. Some 8 percent of the survivors and 3 percent of the deceased had zero HCE in all 36 months. About one-third of the survivors and two-thirds of the deceased had positive HCE in a given month, again pointing to higher (expected) HCE of the deceased.

[1] HCE are expressed in CHF of 2006

Table 2: Descriptive statistics II, 1997-1999

Month	Variable	Deceased				Survivors			
		Mean	SE	Max	Share of HCE>0	Mean	SE	Max	Share of HCE>0
12/1999	HCE m1	1185	1904	20632	0.67	219	219	682	0.34
11/1999	HCE m2	1287	2156	24420	0.68	286	286	871	0.38
10/1999	HCE m3	1326	2176	26044	0.68	299	299	977	0.35
09/1999	HCE m4	1247	2295	43712	0.65	275	275	818	0.36
08/1999	HCE m5	1264	2198	24486	0.64	255	255	892	0.32
07/1999	HCE m6	1221	2449	52618	0.65	252	252	852	0.34
06/1999	HCE m7	1127	2020	39665	0.66	277	277	843	0.37
05/1999	HCE m8	1141	2253	41951	0.64	251	251	776	0.34
04/1999	HCE m9	1120	2115	56078	0.63	267	267	905	0.34
03/1999	HCE m10	1156	2104	22002	0.66	305	305	960	0.37
02/1999	HCE m11	1006	2248	62890	0.60	246	246	902	0.33
01/1999	HCE m12	1389	2347	23199	0.70	358	358	1043	0.40
12/1998		804	1600	19277	0.57	190	190	704	0.31
11/1998		949	1898	32298	0.60	232	232	748	0.34
10/1999		1015	1970	29759	0.61	265	265	861	0.34
09/1998		929	2118	48687	0.59	253	253	783	0.34
08/1998		865	1963	45309	0.56	222	222	935	0.28
07/1998		915	1919	36943	0.57	233	233	1047	0.30
06/1998		837	1619	21411	0.57	263	263	1018	0.32
05/1998		860	1807	29406	0.58	243	243	919	0.32
04/1998		944	2040	32920	0.57	246	246	852	0.31
03/1998		930	2039	28690	0.57	263	263	886	0.33
02/1998		762	1669	27646	0.52	214	214	766	0.30
01/1998		1247	2311	28059	0.65	332	332	944	0.37
12/1997		633	1678	54217	0.50	174	174	652	0.29
11/1997		676	1539	20575	0.52	205	205	634	0.31
10/1997		795	1627	19985	0.57	248	248	937	0.32
09/1997		738	1737	37988	0.53	243	243	833	0.31
08/1997		712	1694	35524	0.49	209	209	819	0.27
07/1997		755	1694	23159	0.51	210	210	801	0.28
06/1997		639	1534	23032	0.51	215	215	907	0.30
05/1997		712	2675	119964	0.50	202	202	678	0.29
04/1997		731	1581	20890	0.54	244	244	833	0.32
03/1997		643	1466	16087	0.51	196	196	645	0.28
02/1997		580	1387	20290	0.48	205	205	710	0.29
01/1997		1048	2144	25797	0.58	306	306	920	0.36
01/1997-12/1999	$\overline{\text{HCE}}_{1\text{-}36}$	950	1022	10952	0.97	247	247	389	0.92

The time series of monthly HCE in *Table 2* reveals a statistical artefact. In January, HCE are markedly higher than in December. This reflects a delay in claims processing by health care providers before Christmas that usually is made up in January. Apart from these peaks, there is a steady increase in the monthly real HCE of the deceased, while they remain relatively stable among the survivors. The steady rise of HCE as time proceeds reflects the shrinking remaining TTD among the deceased, i.e. of persons who were to die between 2000 and 2002.

Model 1 specifies the following equation with time to death observed in January 2000 as the dependent variable,

$$\begin{aligned} TTD_{2000} = {} & \alpha_0 + \sum_{m=1}^{12} \alpha_{1,m}\, ln\left(HCE_{1999,m}\right) + \sum_{m=1}^{12} \alpha_{2,m} \left[ln\left(HCE_{1999,m}\right)\right]^2 \\ & + \alpha_3\, ln\left(\overline{HCE}\right) + \alpha_4 \left[ln\left(\overline{HCE}\right)\right]^2 + \alpha_5 \sum_{m=1}^{12} n_{1999,m} + \alpha_6 \sum_{m=1}^{12} n_{1998,m} \\ & + \alpha_7 Age + \alpha_8 Age^2 + \alpha_9 D + \sum_{j=1}^{5} \alpha_{10,j} E_j + \varepsilon \end{aligned} \tag{1}$$

where $\overline{HCE} = \frac{1}{36} \sum_{t=1997}^{1999} \sum_{m=1}^{12} HCE_{t,m}$ and $n_{t,m} = \begin{cases} 1 & \text{if } HCE_{t,m} > 0 \\ 0 & \text{if } HCE_{t,m} = 0 \end{cases}$.

Explanatory variables include the logarithm of monthly HCE in 1999 and its squared values to reflect recent impacts and to allow for non-constant marginal returns; the average monthly HCE during in the three preceding years (1997 to 1999) and its squared value to reflect lagged influences and to neutralize the January spike found in Table 2, again admitting non-constant marginal returns; the number of months with positive HCE in 1999 and 1998, respectively, to have an indicator of subjective health status (on the premise that this is what determines the decision to initiate a health care episode); age and age squared, survivor status ($D = 1$ if deceased); five dummy variables E_j indicating an individual's canton of residence (Zurich = 1, Geneva = 0) and the four insurance parameters listed in *Table 1*, and an error term. Zero HCE observations were set equal to 1 in order to allow transformation to logarithms.

Table 3 presents the estimation results for eq. (1). Turning first to the estimate including survivor status *D*, one finds that the coefficient for the linear effect of HCE on TTD (α_1) is positive while the coefficient for the squared effect (α_2) is negative. The combined marginal effect of an increase in HCE on TTD is positive for all months of 1999. For the deceased, it is negative in the last month (HCE_{m1}), possibly pointing to the onset of decreasing marginal returns when the time of death is close. For the average monthly HCE between 1997 and 1999, no significant effect on TTD is observable. The number of months with positive HCE in 1999 is significant. However, while a high number of months with positive HCE in 1999 does point to a shorter remaining life expectancy, the reverse is true for 1998. Interestingly, neither age nor sex nor the presence of (other) insurance except supplementary hospital coverage constitute significant predictors. But the fact that the person died during the 36 months to come leads to the prediction that TTD is 17 months less.

Table 3: T**ime to death (TTD) measured at the end of 1999, OLS estimation** corrected for heteroskedasticity

	With survival status		Without survival status	
	(1)	(2)	(3)	(4)
Dep. Var.	**Coeff.**	**Std. err.**	**Coeff.**	**Std. err.**
lnHCE$_{m1}$	0.963**	0.1710	1.433**	0.2352
lnHCE$_{m2}$	0.783**	0.1677	1.370**	0.2275
lnHCE$_{m3}$	0.497**	0.1653	0.905**	0.2311
lnHCE$_{m4}$	0.541**	0.1703	0.991**	0.2293
lnHCE$_{m5}$	0.481**	0.1667	1.030**	0.2300
lnHCE$_{m6}$	0.568**	0.1665	0.886**	0.2268
lnHCE$_{m7}$	0.570**	0.1681	0.915**	0.2297
lnHCE$_{m8}$	0.552**	0.1683	0.959**	0.2313
lnHCE$_{m9}$	0.402*	0.1695	0.817**	0.2333
lnHCE$_{m10}$	0.476**	0.1690	0.811**	0.2336
lnHCE$_{m11}$	0.511**	0.1667	0.696**	0.2239
lnHCE$_{m12}$	0.517**	0.1639	0.910**	0.2231
(lnHCE$_{m1}$)^2	-0.123**	0.0210	-0.184**	0.0285
(lnHCEm2)^2	-0.085**	0.0199	-0.147**	0.0269
(lnHCE$_{m3}$)^2	-0.043*	0.0196	-0.085**	0.0275
(lnHCE$_{m4}$)^2	-0.053**	0.0206	-0.096**	0.0276
(lnHCE$_{m5}$)^2	-0.042*	0.0199	-0.111**	0.0274
(lnHCE$_{m6}$)^2	-0.046*	0.0198	-0.078**	0.0267
(lnHCE$_{m7}$)^2	-0.054**	0.0199	-0.079**	0.0274
(lnHCE$_{m8}$)^2	-0.047*	0.0203	-0.081**	0.0282
(lnHCE$_{m9}$)^2	-0.033	0.0200	-0.069*	0.0276
(lnHCE$_{m10}$)^2	-0.034	0.0199	-0.059*	0.0276
(lnHCE$_{m11}$)^2	-0.044*	0.0198	-0.044	0.0263
(lnHCE$_{m12}$)^2	-0.037	0.0190	-0.070**	0.0258
$ln\,\overline{HCE}$	0.001	0.1292	0.988**	0.1933
$(ln\,\overline{HCE})^2$	-0.002	0.0247	-0.213**	0.0348
n_99	-1.535**	0.3813	-2.581**	0.5221
n_98	0.084*	0.0388	0.173**	0.0530
Age	0.036	0.0290	0.297**	0.0375
age^2	0.000	0.0003	-0.004**	0.0003
Sex$_m$	-0.248	0.1427	-2.222**	0.1984
Zurich	-0.029	0.1695	-0.703**	0.2412
higher deductibles	0.021	0.1313	0.636**	0.1955
accident insurance	0.015	0.1179	-0.566**	0.1914
suppl. hospital coverage	0.042	0.1371	0.529**	0.1946
other supplements	0.504	0.2879	1.419**	0.3983
survivor status	-16.768**	0.2300		
Constant	34.664**	0.8399	29.647**	1.1129
R^2	0.670		**R^2**	0.352
Root MSE	6.121		**Root MSE**	8.5722
F (37, 8612)	271.56		**F (36, 8613)**	121.48
N	8,650		**N**	8,650

Things change considerably when survival status is excluded (columns 3 and 4 of Table 3). The combined positive marginal effect of monthly HCE during 1999 is now reinforced and the marginal effect of average HCE during the last 36 months becomes significantly positive as well. Age, gender, and (other) insurance coverage become significant, with the expected signs. Finally the coefficient of determination drops 67 to 35 percent.

Since survival status cannot be known in a forecast, it is this second version of Model 1 that will be used to calculate the expected TTD of individuals at the end of 2002 (our instrument for the IV estimation in model 2), based on monthly HCE between 2000 and 2002 and the other explanatory variables.

3. Testing for the endogeneity of time-to-death

The second step consists in estimating HCE of 2003,

$$HCE_{2003} = \beta_0 + X\beta_1 + \beta_2 TTD + \eta \ , \tag{2}$$

where X is a $1\times L$ vector of exogenous explanatory variables, β_1 is a $L\times 1$ coefficient vector, and η an error term. For simplicity, eq. (2) denotes both elements of the two-part model. As shown in the preceding section, TTD is endogenous to HCE, implying that $Cov(TTD,\eta) \neq 0$, causing coefficient estimates to be biased and inconsistent.[2]

The instrumental variable (IV) approach calls for replacing observed *TTD* by an estimate provided by an auxiliary regression that contains at least one variable *z*, which is not part of eq. (2). Such an estimate must meet the following requirements [Wooldridge (2009, ch. 15.1)]: (i) it should be highly correlated with the endogenous variable, and (ii) it must not be correlated with the error term in (2). If condition (i) is violated, the equation designed to derive 'purged' values for the endogenous regressor is likely misspecified, imparting a risk of inconsistency to the estimation of eq. (2). If condition (ii) is violated, estimation of (2) is inconsistent with certainty. Since the regressors *X* listed in Table 1 are assumed to be exogenous, they belong to the reduced form given by

$$TTD = \gamma_0 + X\gamma_1 + Z\gamma_2 + u \ . \tag{3}$$

Here, the vector *Z* contains the instruments. By construction, the TTD values derived from eq. (1) qualify as instruments, such that

$$z_1 = \widehat{TTD_i}(\hat{\alpha}) \ , \tag{4}$$

Here, $\hat{\alpha}$ denotes the subset of estimated coefficients pertaining to the 12 lagged monthly HCE terms and their squared values and the other variables from eq. (1). Further candidates

[2] Eq. (2) could include survival status (*D*) as well. This variable is likely also plagued by endogeneity. However, this problem will be neglected because there is a lack of instruments satisfying the conditions stated in the text. Moreover, including survival status as an exogenous variable does not produce reasonable IV-estimates, possibly due to the endogeneity of survival.

for instruments would be average monthly HCE during the past three years and the insurance variables comprising the vector *E* in eq. (1).

Requirement (i) for valid instruments in the case of endogeneity can simply be tested by using regression (3). If $\gamma_2 \neq 0$ holds, requirement (i) is met. Requirement (ii) calls for $\rho_2 = 0$ in the regression,

$$\hat{\eta} = \rho_0 + X\rho_1 + Z\rho_2 + \varpi. \tag{5}$$

The $\hat{\eta}$ are the residuals calculated from eq. (2), and they should be uncorrelated with the instruments contained in *Z*. However, since there are more than one possible instrument for the one endogenous variable *TTD*, testing for the so-called overidentifying restriction is possible, indicating whether the excluded instruments are correctly excluded from eq. (5). This test can be performed using the $\chi^2(Q)$ distribution, with *Q* denoting the number of instruments. The test statistic is equal to NR^2, with R^2 estimated from (5) and *N*, the number of observations (Wooldridge 2002, p. 122).

Once the set of valid instrument is established, we can test the exogeneity of TTD via a regression-based Hausman test which employs the estimated error term of the reduced equation ($\hat{u}$) (see equation (3)) as an additional explanatory variable in the structural form of the model (equation (2)). The null hypothesis of exogeneity of TTD can be tested by a simple *t*-test on the coefficients of $\hat{u}$. Wooldridge (2002, p. 474) shows that this procedure is also appropriate in the case of Probit estimation.

Eq. (2) was estimated as a two-part model, relating annual HCE of 2003 to TTD measured up to the end of 2006, with its maximum value again set at 36 months. Results for the two-part in *Table 4* refer to the probability of incurring positive HCE.[3] 'While details of the reduced form equation (3) and the test equation (5) are not shown, 'accident insurance' (see *Table 1*) appears to be a valid instrument in addition to estimated *TTD*. In particular, both instruments passed the test for the overidentifying restrictions (see the right-hand side of *Table 4*). However, according to the Hausman test, one has to reject the null hypothesis of exogeneity for *TTD*, as evidenced by the $\chi^2(1)$ test statistic (again on the right-hand side of *Table 4*).

This might be due to autocorrelation in HCE. In that case, $\widehat{TTD}$ would pass the test eq. (5) which is based on contemporaneous values. Yet, through eq. (4) its values still would contain

[3] Actually, the threshold is set at CHF 230 rather than zero. The minimum annual deductible was CHF 230 at the time, preventing the insured from submitting bills below that threshold.

error components of previous HCE, which would cause it to correlate with the error term η of eq. (2). Without pursuing this further, the conclusion is that, the two instruments retained, $\widehat{TTD}$ and 'accident insurance', are not fully valid.

Table 4: Probit estimation for HCE > 230 CHF, 2003

	Probit		IV Probit	
	(1)	(2)	(3)	(4)
	Coeff.	Robust std. err.	Coeff.	Robust std. err.
TTD	-0.018**	0.002	-0.030**	0.003
Age	-0.155**	0.019	-0.167**	0.019
Age^2/1000	2.913**	0.341	3.167**	0.336
Age^3/1000	0.015**	0.002	-0.017**	0.002
Sex_m	-0.932**	0.058	-0.918**	0.058
Sex_m*Age	0.010**	0.001	0.010**	0.001
Zurich	-0.329**	0.021	-0.329**	0.021
Higher deductible	-0.500**	0.014	-0.497**	0.014
Suppl. hospital cov.	0.121**	0.015	0.122**	0.015
Other supplements	0.297**	0.026	0.303**	0.026
Constant	3.948**	0.366	4.575**	0.373
Number of observations		46,299	46,299	
Wald χ^2 (10)	5.249		**Wald χ^2 (10)**	5,593
Pseudo R-squared	0.129		**Hausman test of exogeneity**	
			χ^2 (1) = 66.49 (p = 0.001)	
			Test of overidentifying restrictions:	
			χ^2 (1) = 2.92 (p = 0.09)	

Note that controlling for endogeneity of TTD is important, its coefficient changing from -0.18 in the Probit estimation to -0.30 in the IV estimation. This result also arises if the second instrument 'accident insurance' is dropped from the equation. Therefore, the effect of TTD on HCE is even reinforced rather than weakened, contrary to expectations. All the other coefficients prove rather robust.

Table 5 reports on the IV regression of positive HCE. Here, no set of instruments satisfying both validity requirements was found. With $\widehat{TTD}$ only, the Hausman test rejects the null hypothesis of exogeneity. Since eq. (3) is now just identified, testing for overidentifying restrictions coming from additional instruments is not possible. However, the transition to IV estimation has a similar effect as in the Probit part of the model in that the coefficient at *TTD* increases in absolute value. The results thus are somewhat inconclusive. While exogeneity of TTD is rejected and instruments are "strong"[4], the instruments used for purging TTD of its

[4] The weakness of instruments can be tested by the so called concentration parameter – a test of instruments in the reduced equation (Stock et al. 2002).

endogeneity are possibly not valid. If endogeneity is not controlled for, the effect of TTD on HCE is underestimated. However, the appropriate tests reveal that it is extremely difficult to find valid instruments. This hints to unobserved variables influencing the error terms in eq. (2), such as morbidity indicators.

Table 5: OLS and IV estimation for ln HCE | HCE > 230 CHF, 2003

	OLS		IV	
	(1)	(2)	(3)	(4)
	Coeff.	Robust std. err.	Coeff.	Robust std. err.
TTD	-0.041**	0.0013	-0.067**	0.0017
Age	-0.010**	0.0036	0.002	0.0036
Age^2/1000	0.223**	0.0291	0.107**	0.0298
Sex_m	-0.660**	0.1887	-0.644*	0.1921
Sex_m*Age	0.025**	0.0064	0.025**	0.0065
Sex_m*Age^2/1000	-0.231**	0.0525	-0.236**	0.0540
Zurich	-0.413**	0.0143	-0.418**	0.0144
Accident insurance	0.213**	0.0144	0.219**	0.0144
Higher deductible	-0.181**	0.0118	-0.172**	0.0118
Suppl. hospital cov.	0.034**	0.0119	0.039**	0.0120
Other supplements	-0.087**	0.0255	-0.070**	0.0258
Constant	9.374**	0.1091	10.021**	0.1146
Number of obs.		35,593	**Number of obs.**	35,593
F (13, 35579)		650	**F (13, 35579)**	639
Prob > F		0	**Prob > F**	0
R-squared		0.1716	**Centered squared**	0.167
Root MSE		1.0621	**Root MSE**	1.06
		0.129	**Hausman test of exogeneity**	
			χ^2 (1) = 28.45 (p = 0.00)	

The non-IV estimation results regarding the age effects confirm previous findings (see *Zweifel* et al., 2004): although the coefficients pertaining to age are significant, the total marginal effect of age is small. Male have significantly lower HCE but the difference decreases with increasing age. In the IV estimation, the marginal effect of age becomes even smaller. The significant impact of TTD on HCE is reinforced, vindicating the findings of the 'red herring' literature.

4. Explaining health care expenditure with expected time to death

Past HCE, sex, age and characteristic of the insurance contract can explain as much as 35 percent of an individual's TTD (see col. 3 in *Table 3*). Therefore, TTD may have little explanatory power over and above past HCE. This issue is explored using model 2 (see

Figure 1 again), which relates HCE of 2003 to estimated *TTD*. The $\widehat{TTD}$ values come from eq. (1), with the period of observation moved forward from 2000 to 2002 and $\overline{HCE}$ modified accordingly to be defined over the preceding 36 months. *Table 6* contains the estimates for the Probit part. Comparing col. 1 with col. 1 of *Table 4* shows coefficients to be robust despite the fact that $ln\,\overline{HCE}$ and its square enter as regressors there. Remarkably, the explanatory power increases from 12.9 percent in *Table 4* to 36.8 percent when observed *TTD* is replaced by $\widehat{TTD}$, derived from the variant of model 1 that includes past monthly *HCE* but excludes the individual's survivor status. Most importantly however, $\widehat{TTD}$ continues to be highly significant.

Table 6: Two-part model with $\widehat{TTD}$ (2003)

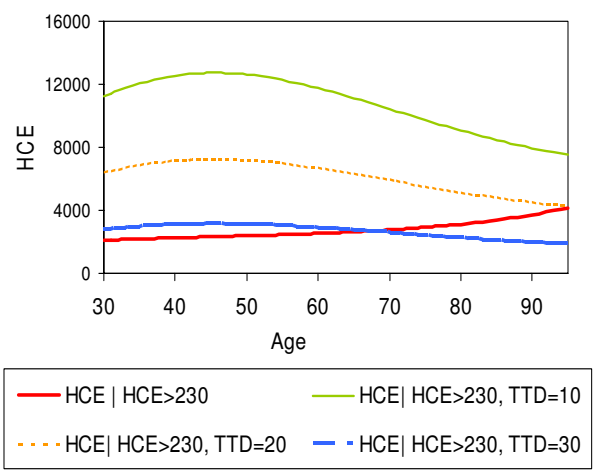
	Probit (HCE > 230)		OLS ln(HCE) | HCE > 230	
	(1)	(2)	(3)	(4)
	Coeff.	SE	Coeff.	SE
$\widehat{TTD}$	0.040**	0.011	-0.085**	0.004
Age	-0.008	0.006	0.037**	0.013
Age^2	0.216**	0.059	-0.483**	0.212
Age^3/1000			0.001**	0.001
Sex$_m$	-0.232**	0.073	-0.129**	0.043
Sex$_m$*Age	0.003*	0.001	0.000**	0.001
Zurich	-0.037	0.027	-0.214**	0.012
Accident insurance	-0.007	0.019	0.001**	0.012
Higher deductibles	-0.313**	0.018	0.008**	0.010
Suppl. hosp. cover.	0.043*	0.018	0.047**	0.010
Other supplements	0.142**	0.036	0.064**	0.021
$ln\,\overline{HCE}$	0.071**	0.021	-0.311**	0.018
$(ln\,\overline{HCE})^2$	0.065**	0.004	0.074**	0.002
Constant	0.065**	0.340	9.427**	0.270
No. of obs.	46,299		**No. of obs.**	35,593
Wald chi2 (13)	11,446		**Wald chi2 (13)**	2,936
Pseudo R^2	0.368		**Pseudo R^2**	0.472
			Root MSE	0.850

As to the conditional HCE part, a comparison of cols. 3 of *Table 4* and *Table 6* again suggests a great deal of robustness. Also note that the explanatory power of the two estimations markedly increases once more when observed *TTD* is replaced by $\widehat{TTD}$. When average monthly *HCE* between 2000 and 2002 is included as explanatory variable for HCE in 2003, $\widehat{TTD}$ keeps its high statistical significance while the model now explains almost 50 percent of the variance of annual HCE. This is substantially higher than what the pertinent literature

reports; *Newhouse* et al. (1989) found that due to the immanent randomness of demand, the maximum R^2 for explaining HCE is 5% for inpatient and 25% for outpatient care.

Having established the explanatory power of $\widehat{TTD}$ over and above past HCE, one still needs to check the robustness of core 'red herring' findings with regard to the irrelevance of age as a predictor of HCE. In *Figure 2*, the age profile of men's conditional positive HCE is illustrated for different values of $\widehat{TTD}$. For $\widehat{TTD}$ = 30, the profile is almost horizontal up to the age of 60 years. Beyond that age, HCE slowly decreases. For $\widehat{TTD}$ = 20, the age profile is falling beyond the age of 50, and for $\widehat{TTD}$ =10, the negative age gradient sets in at age 45. This pattern may be interpreted in the following way. Assume that treating physicians and/or their patients can predict time to death to the extent that they are able to distinguish between 10, 20, and 30 months remaining. Then, $\widehat{TTD}$ = 10 clearly indicates that chances of survival are relatively low, causing patients to give up pretty much regardless of age, i.e. at an early age such as 45. This causes HCE to stabilize at that age. By way of contrast, $\widehat{TTD}$ = 30 is a much less informative signal because ex ante there are still many chances of recovery. This means that it pays to fight up to a higher age such as 60, after which these efforts (and with them, HCE) taper off.

Figure 2: Age profile of conditional positive HCE for different expected TTD



Compared to these small, second-order age effects in *Figure 2*, estimated proximity to death is far more important. HCE at $\widehat{TTD}$ = 10 is almost double the peak value of HCE at

$\widehat{TTD}$ = 20, which in turn almost doubles the peak value at $\widehat{TTD}$ = 30. Therefore, *Figure 2* replicates two well-known properties of the cost of dying. First, they are large compared to any age effects, and second, they are decreasing beyond the age of 60 at the latest (*Lubitz* and *Riley*, 1993, *Felder* et al., 2000, *Schellhorn* et al., 2000, *Chernichowski* and *Markowitz*, 2004). This may be contrasted with the "naïve" approach that fails to control for time to death, represented by the curve that shows the lowest HCE up to age 65 but then crosses the other curves, steadily increasing throughout the life cycle.

5. Conclusion

This paper is devoted to an issue that has been undermining the credibility of the 'red herring' hypothesis, viz. that time-to-death (TTD) rather than age is a crucial determinant of individual health care expenditure (HCE). If HCE is effective, it should push the time of death away, thus increasing TTD. This would likely result in an overestimate of the effect of TTD on HCE. In model 1, monthly observations on HCE from 1997 to 1999 and individual and insurance contract characteristics are used to explain TTD measured from 2000 to the end of 2002, with its maximum set at 36 months. Past HCE is found to have a positive effect on TTD except for the last month prior to death. Next, values of TTD estimated from model 1 might serve to replace observed values when it comes to explaining HCE of the year 2003. However, they fail a Hausman exogeneity test. Therefore, an Instrumental Variable estimation of model 2 which relates HCE of 2003 to the same variables shifted forward by three years must be imperfect. Still, estimated TTD retains its explanatory power in both components of a two-part model even when past HCE and its square enter as explanatory variables. Moreover, model 2 explains almost 50 percent of the variance in individual HCE, far more than reported in previous studies. Finally, the implied age gradient of HCE is flat, as predicted by the 'red herring' hypothesis.

On the whole, while it proved impossible to fully purge TTD of its endogeneity, the empirical evidence supports the core claim of the 'red herring' hypothesis. Therefore, upward shifts in HCE over time are much more likely caused by advances in medical technology rather than ageing of the population. These advances, if applied to the aged or even the deathbound, could also be responsible for the so-called steepening of the age profile of HCE over time. Unfortunately, the data do not permit to investigate this intriguing conjecture.

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